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JANUARY, 1947

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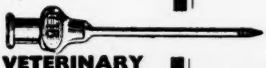
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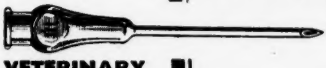
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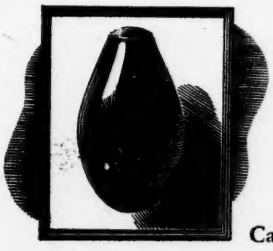

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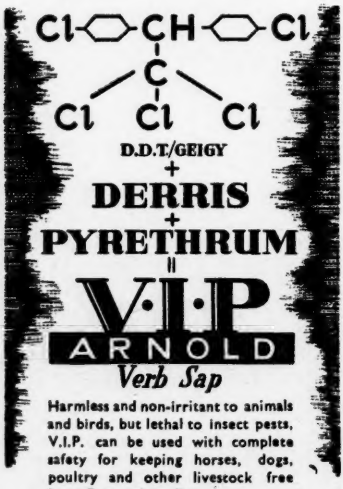

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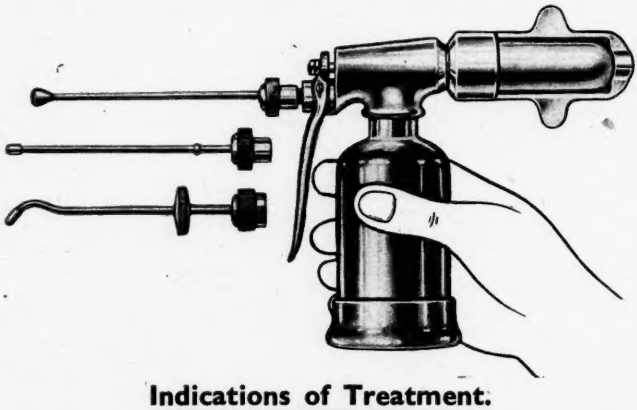

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Vol. 103 January, 1947 No. 1

EDITORIAL

THE NEW YEAR

By the time this issue of the Veterinary Journal is in the hands of the reader, we will be in the year 1947. We take this opportunity of sending to our readers our good wishes for 1947 with the hope that before another year has passed the world will have settled down considerably and that signs of peaceful times will be evident. The life of the world is gradually changing as from war-time to peace-time conditions: changes have to be gradual in order to be lasting. With the necessary patience and endeavour which each of us is capable of practising, we will reach that state of happiness and content which makes for peace and goodwill among all peoples.

All enterprises have passed through troublous times, and more settled conditions are beginning to show themselves; it will be the strong endeavour of those associated with the Veterinary Journal to continue to present to its readers the results of work, clinical and research, which will keep them up to date and enable them, we hope, to put into practise the newest and most satisfactory methods for the control of the afflictions of our domesticated animals and the maintenance and improvement of the health of our livestock.

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GENERAL ARTICLES

OBSERVATIONS ON SOME METABOLIC DISORDERS OF COWS, AS EVIDENCED BY CHEMICAL ANALYSIS OF SAMPLES OF BLOOD FROM CLINICAL CASES

(1) Conditions of Occurrence

By W. M. ALLCROFT

Ministry of Agriculture, Veterinary Laboratory, Weybridge

Introduction

In 1933, on the opening of a small biochemical section of this laboratory, under Dr. H. H. Green, analyses of blood samples submitted by veterinary practitioners were commenced as an aid to, or in confirmation of, clinical diagnosis of metabolic disorders of farm animals. This paper covers miscellaneous data over almost seven years, from 1934 to 1941, and is based on the chemical analyses carried out and on the clinical reports submitted in connection with over 3,000 cases sent from all over the British Isles and from Jersey. Every sample included in this study was taken from an animal when first visited and before any treatment had been carried out, all subsequent samples being omitted.

Literature.—Much work has been published on metabolic disorders of cows, but it is not proposed to attempt any complete bibliography at this juncture or to discuss more than a few of the papers.

Classification.—The metabolic disorders discussed in this paper are classified according to the chemical analyses of the blood samples and not on the basis of clinical diagnosis. Each biochemical type is discussed without relation to the clinical reports which accompanied the samples; thus the term acetonæmia refers to the chemical level of ketone bodies in the blood serum and not to any clinical syndrome, while the term hypomagnesæmia is used irrespective of hyperæsthesia.

Three of the veterinary practitioners, who between them submitted a very large proportion of the total material sent to the laboratory, have already discussed the chemical and clinical aspects of their own cases. [Barker (1939), Weighton (1942) and Messervy (1943).] In these three papers the earlier chemical classification of acetonæmia adopted by Weybridge was used by the authors. Study of the full data available and of the work of Eden and Green (1940) have caused this classification to be revised, and this should be remembered when comparing the results of this paper with those of the above-mentioned papers.

This alteration of the classification of acetonæmia and the resultant distribution of numbers of cases is shown in Table I.

TABLE I

COMPARISON OF THE OLD AND NEW CLASSIFICATION OF ACETONÆMIA

	Old classification		*New classification*	
Designation (*a*)	Serum ketones mg. p.c. (*b*)	Number of cases (*c*)	Serum ketones mg. p.c. (*d*)	Number of cases (*e*)
Normal range of ketones	0.0 to 5.0	—	0.0 to 10.0	—
Mild acetonæmia	5.1 to 15.0	347	10.1 to 20.0	162
Marked acetonæmia	15.1 and above	380	20.1 and above	271

The biochemical types of disorder, which are discussed herein, together with the commonly used clinical terminology, are shown in Table II.

TABLE II

BIOCHEMICAL TYPES OF DISORDER AND THEIR CLINICAL TERMINOLOGY

Biochemical type	Clinical terminology
Hypocalcæmia with normal magnesium Hypocalcæmia with hypermagnesæmia	Milk fever, or post-parturient paralysis
Hypomagnesæmia with normal calcium Hypomagnesæmia with hypocalcæmia	Lactation tetany, grass tetany or atypical milk fever
Mild acetonæmia, marked acetonæmia, with or without hypoglycæmia	Acetonæmia, post-parturient dyspepsia, or Stinson's disease

Throughout this paper the terms employed to denote a high or low blood level are defined as follows:—

Hypocalcæmia:	Values of serum calcium ...	8.4 mg. p.c. and	below
Hypomagnesæmia:	„ „ „ magnesium ..	1.7 „ „	„
Hypermagnesæmia:	„ „ „ „ ..	3.3 „ „	above
Hypoglycæmia:	„ „ blood sugar ...	40.0 „ „	below
Hyperglycæmia:	„ „ „ „ ...	71.0 „ „	above
Acetonæmia:	See new classification in Table I, column (*d*).		

Chemical Methods

(i) Serum calcium and magnesium were determined on the same sample of serum, using the Clark and Collip (1925) modification of the method of Kramer and Tisdall (1921) for calcium, and the method of Denis (1922) for magnesium. A microsiphon technique, described by Salt (1935), was used for the transfer of the supernatant fluid for magnesium estimation and for removal of the wash fluids after each centrifugal washing of the precipitates. Three washings were used for the magnesium ammonium phosphate precipitate. Checks of a standard solution containing 10 mg. calcium and 2 mg. magnesium per 100 ml. were regularly run through with every batch of sera to ensure accuracy of manipulative technique.

(ii) Serum ketones, as acetone, were determined by the method quoted by Eden and Green (1940).

(iii) Blood sugar was determined by the method of Hagedorn and Jensen (1920), using the modification of Somogyi (1930) for protein precipitation.

(iv) Inorganic P was determined by the colorimetric method of Fiske and Subbarrow (1925), which was also used for the final steps of magnesium estimation.

(v) Hæmoglobin was determined by the colorimetric method of Newcomer (1923), using a disc standardised against acid hæmatin solutions.

Results

The metabolic disorders under consideration (Table II) are assembled in relation to seasonal occurrence, the number of times the cow had calved and the time interval since her last calving. In the case of seasonal occurrence, an attempt has been made to show the different seasonal incidence of the disorders in Britain

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TABLE III

SEASONAL INCIDENCE OF CERTAIN TYPES OF BIOCHEMICAL DISORDER IN JERSEY AND THE BRITISH ISLES

NUMBER OF CASES

Month of the year	JERSEY				BRITISH ISLES				ALL SOURCES	
	Marked aceto-næmia	Mild aceto-næmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with hyper-mag-nesæmia	Marked aceto-næmia	Mild aceto-næmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with hyper-mag-nesæmia	Hypo-calcæmia with hypo-mag-nesæmia	Hypo-mag-nesæmia with normal calcium
(*a*)	(*b*)	(*c*)	(*d*)	(*e*)	(*f*)	(*g*)	(*h*)	(*i*)	(*j*)	(*k*)
September	8		1	1	4	2	24	2	3	1
October	10	3	5	3	3		23	6	8	9
November	16	7	9	14	7	3	21	9	18	4
December	19	4	10	10	13	12	41	5	47	15
January	11	8	14	11	12	7	22	4	37	8
February	14	3	8	19	16	11	37	11	31	10
March	21	3	12	24	19	9	48	8	48	19
April	26	7	19	28	14	3	31	7	62	19
May	18	3	12	22	3	4	66	14	41	4
June	38	7	13	23	1	5	58	14	11	2
July	33		14	14	1	2	44	13		2
August	12		1	1	5	6	52	13	4	4
TOTAL	226	45	118	170	98	64	467	106	310	96

PERCENTAGE OF CASES

Month of the year	JERSEY				BRITISH ISLES				ALL SOURCES	
	Marked aceto-næmia	Mild aceto-næmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with hyper-mag-nesæmia	Marked aceto-næmia	Mild aceto-næmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with hyper-mag-nesæmia	Hypo-calcæmia with hypo-mag-nesæmia	Hypo-mag-nesæmia with normal calcium
(*a*)	(*b*)	(*c*)	(*d*)	(*e*)	(*f*)	(*g*)	(*h*)	(*i*)	(*j*)	(*k*)
September	3.5		0.8	0.6	4.0	3.1	5.1	1.9	1.0	1.0
October	4.4	6.7	4.2	1.8	3.0		4.9	5.7	2.6	9.4
November	7.1	15.6	7.6	8.2	7.1	4.7	4.5	8.5	5.8	4.2
December	8.4	8.9	8.4	5.9	13.2	18.8	8.8	4.7	15.1	15.5
January	4.9	17.5	11.9	6.5	12.2	10.9	4.7	3.8	11.9	8.3
February	6.2	6.7	6.8	11.2	16.4	17.2	7.9	10.3	10.0	10.4
March	9.3	6.7	10.2	14.0	19.6	14.1	10.3	7.5	15.6	19.8
April	11.5	15.6	16.2	16.5	14.3	4.7	6.6	6.6	20.0	19.8
May	8.0	6.7	10.2	13.0	3.0	6.2	14.2	13.2	13.2	4.2
June	16.8	15.6	11.0	13.5	1.0	7.8	12.5	13.2	3.5	2.1
July	14.6		11.9	8.2	1.0	3.1	9.4	12.3		2.1
August	5.3		0.8	0.6	5.1	9.4	11.1	12.3	1.3	4.2
TOTAL	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

and Jersey. The total number of cases and their percentage distribution are shown in tabular form and the histograms of percentage distribution are also given for ready comparison.

Seasonal occurrence in Britain and Jersey

The relevant data are presented in Table III and Fig. 1. In compiling these data, the totals recorded for each calendar month are the totals for six years. For the mineral disturbances, the period covers January, 1934, to December, 1940, inclusive, while the acetonæmia cases occurred over the period September, 1935, to August, 1941, inclusive, as ketone determinations were not made in the earlier days. It is felt that this presentation, though not without its limitations, gives a fair index of the likely seasons of occurrence of the different disorders.

Marked acetonæmia occurred fairly generally throughout the year in Jersey, with a slightly higher incidence in June and July, when 31.4 per cent. of cases were recorded. In the British Isles there appeared to be a seasonal occurrence, with the bulk of cases in the winter period, December to April, and only a few sporadic cases over the remaining seven months. Though the cases of mild acetonæmia were fewer, the seasonal distribution, in both localities, was similar to that of marked acetonæmia.

Hypocalcæmia, with normal or high values for magnesium, showed differential occurrence in Britain and Jersey. It was noted that of the 288 cases recorded for Jersey, 59 per cent. showed hypermagnesæmia and 41 per cent. normal magnesium values. Of the 573 cases for Britain, however, 18.5 per cent. showed hypermagnesæmia and 81.5 per cent. normal magnesium values. This at first suggested a breed difference, with a tendency to hypermagnesæmia in Jersey, or even Channel Island, breeds. That this difference is not genuine, however, is shown by the comparison of breeds in Britain, shown in Table IV, from which it is at once clear that under British conditions the combination of lowered calcium and elevated magnesium occurs in animals of many breeds, even in a beef cow.

TABLE IV

DISTRIBUTION OF BREEDS IN 106 CASES OF HYPOCALCÆMIA AND HYPERMAGNESÆMIA OCCURRING IN THE BRITISH ISLES

Breed	Number of cases
Jersey	17
Guernsey	4
Dairy Shorthorn	45
Holstein	6
Ayrshire	5
Dexter	5
Hereford	1
Crossbred	18
Unspecified	5
	106

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In considering the cases of typical hypocalcæmia, all cases of marked acetonæmia have been omitted, as there is a tendency for many such cases to show also some measure of hypocalcæmia.

In Britain, the seasonal occurrence of hypocalcæmia was much the same whether the magnesium was normal or high. Cases occurred all the year round, but 50 per cent. occurred in the four months May to August inclusive. In Jersey, there was a difference according to the level of magnesium; when magnesium was normal, there were few cases between August and October and the bulk of cases were evenly distributed over the remaining nine months, while of those cases showing hypermagnesæmia, 76.4 per cent. of the cases occurred over the six months February to July inclusive.

In considering hypomagnesæmic cases, only those for Britain are considered as the incidence of this condition is very low in Jersey. When the serum calcium was also low, 85.8 per cent. of cases occurred between December and May, with highest monthly incidence in December and April. When calcium was normal, 72.8 per cent. occurred between December and April, with March and April as the months of highest incidence.

Occurrence in Relation to Number of Calvings

Since these disorders are largely associated with the functions of reproduction and lactation, the number of calvings was considered a better criterion than the age in years. The data on this aspect of occurrence are assembled in Table V and shown in Figure 2.

Out of 1,429 cases on which the relevant information is available, only nine cases were in pregnant heifers; of those, seven showed hypomagnesæmia with normal calcium. In first calvers, the general incidence was low, except in cases of marked acetonæmia, when 52, or 18.8 per cent., of the cases occurred.

Marked acetonæmia was commonest in first calvers, 18.8 per cent., and third, fourth and fifth calvers, 51.6 per cent. of all cases recorded. Mild acetonæmia was commonest in third to sixth calvers, 68.3 per cent. of cases.

Hypocalcæmia with normal magnesium was commonest in third to sixth calvers, 72.7 per cent., while fourth calvers accounted for 25 per cent. of all cases. If the magnesium was high the incidence of cases was greatest in fourth to seventh calvers, 70 per cent., with 45.7 per cent. in fourth and fifth calvers.

Hypomagnesæmia was associated with earlier calvings. When accompanied by hypocalcæmia, 44.2 per cent. of cases were third and fourth calvers, and 30.6 per cent. fifth and sixth calvers. When the calcium was normal, 28.6 per cent. were pregnant heifers or first and second calvers, and a further 41.6 per cent. in third and fourth calvers.

Occurrence in Relation to the last Date of Calving

In presenting these data, the time interval after calving has been based on the subdivision of the reproductive cycle given by Barker (1939). The first four days post-partum form the puerperium, the next four the interregnum, then follows an indefinite period of anœstrus and a sequence of œstrus and diœstrus until pregnancy again commences.

The available data are given in Table VI and Figure 3.

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TABLE V

ASSOCIATION OF CERTAIN TYPES OF BIOCHEMICAL DISORDER WITH THE NUMBER OF TIMES THE COW HAS CALVED

Number of calvings	ACTUAL NUMBER OF SAMPLES								PERCENTAGE DISTRIBUTION OF SAMPLES						
	Marked acetonæmia	Mild acetonæmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with Hyper-magnesæmia	Hypo-calcæmia with Hypo-magnesæmia	Hypo-magnesæmia with normal calcium	All cases		Marked acetonæmia	Mild acetonæmia	Hypo-calcæmia with normal magnesium	Hypo-calcæmia with hyper-magnesæmia	Hypo-calcæmia with hypo-magnesæmia	Hypo-magnesæmia with normal calcium	All cases
0		1		1		7	9	...		1.2		0.4		9.1	0.6
1	52	2	3	2	6	6	71	...	18.8	2.3	0.5	0.9	2.7	7.8	4.9
2	15	8	12	4	17	9	65	...	5.4	9.4	2.2	1.9	7.7	11.7	4.5
3	49	12	74	14	43	18	210	...	17.8	14.1	13.3	6.5	19.4	23.4	14.7
4	55	19	138	47	55	14	328	...	20.0	22.5	25.0	21.9	24.8	18.2	23.1
5	38	17	98	51	33	8	245	...	13.8	20.0	17.7	23.8	14.9	10.4	17.3
6	17	10	93	30	35	4	189	...	6.1	11.7	16.7	14.0	15.7	5.2	13.2
7	19	5	48	22	11	3	108	...	6.9	5.9	8.6	10.3	4.8	3.9	7.5
8	13	7	43	17	7	2	89	...	4.7	8.2	7.7	7.9	3.2	2.6	6.2
9	2	1	14	12	2	1	32	...	0.7	1.2	2.5	5.6	0.9	1.3	2.2
10	7	3	15	10	7	3	45	...	2.5	3.5	2.7	4.6	3.2	3.9	3.1
Over 10	9		17	4	6	2	38	...	3.3		3.1	1.9	2.7	2.6	2.7
								...							
TOTAL	276	85	555	214	222	77	1,429	...	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Marked acetonæmia occurred most commonly in the third week after calving, 25.9 per cent. of cases, while the period ninth to forty-second day post-partum included 71.6 per cent. of cases. Of the remainder, 10.9 per cent. were in the puerperium and 7.1 per cent. in the interregnum.

Mild acetonæmia cases showed 54.2 per cent. in the puerperium and 10.6 per cent. in the interregnum.

Hypocalcæmia, with either normal or high magnesium, was commonest in the puerperium, 86.5 and 85.0 per cent. respectively, while the incidence in the first two days post-partum was 80.0 and 82.7 per cent. respectively.

When hypomagnesæmia was observed, whether the calcium was normal or low, there was no association with the interval since last calving, except that 36.2 per cent. of cases showing hypocalcæmia occurred in the puerperium.

Discussion of Results

These data, acquired over a long period, are presented in concise form, as the conclusions to be drawn were considered to be of general interest. It is appreciated that in the absence of accurate statistics for calvings in the areas from which material has been received, the relative incidence of different types of cases cannot be really assessed, but the results recorded show definite tendencies which are of importance in animal husbandry.

Marked Acetonæmia

Acetonæmia is a chemical symptom of a disturbance of the fat and carbohydrate metabolism of the animal, and the mass of published work suggests that it may arise from one or more of many causes. It is shown that there is a difference in the seasonal incidence of this condition in Britain and in Jersey, and this difference is striking. The greatest incidence occurs in the period two to six weeks post-partum and might, therefore, be expected to show association with the general calving seasons, on which full data are not available. There is also a difference in the husbandry of dairy cattle in Jersey and in Britain. Messervy (1943) refers to the general practice of Jersey, which he records as turning cows out by day and in by night all the year round. It is also known that many of the cows in Jersey are tethered at pasture, reducing their activity. In Britain, there is no hard and fast system of husbandry, but it is general to stall-feed dairy cows in the winter and to turn them out to grass part or all of the day in summer. That access to pasture had little effect on the incidence of acetonæmia was shown by examining the records of acetonæmia cases of which the feeding records were also available. Between December and April, 33 cases had access to pasture and 26 were stall-fed with no access to pasture. Between May and November, 11 cases were at pasture and four had no access to pasture.

This disorder was the only one studied which had any marked incidence in first calvers, while the third to fifth calvings were the most likely time of attack.

Mild Acetonæmia

This condition is one which is seldom recognised clinically and the significance of which is uncertain. Its seasonal occurrence was found to be roughly the same

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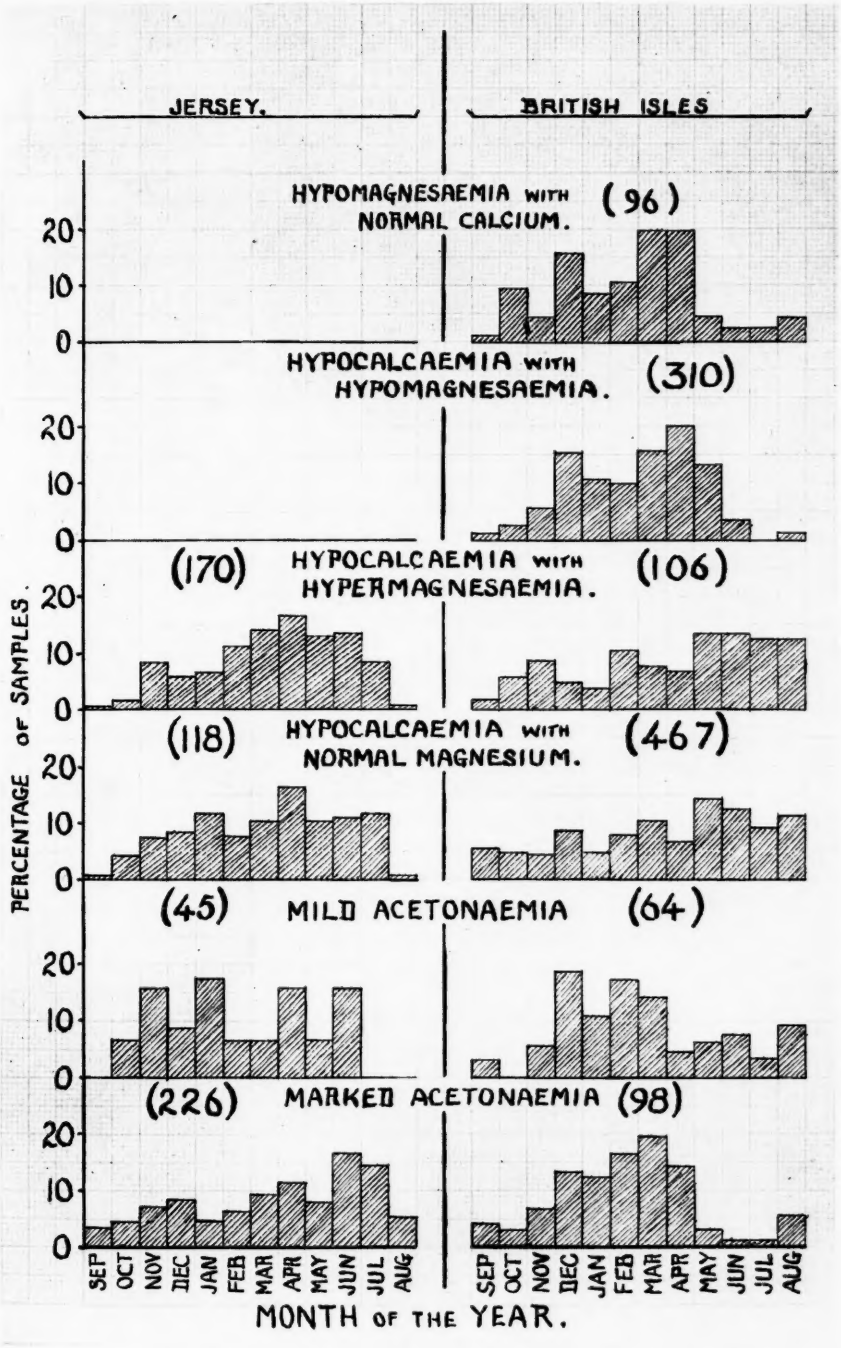

FIG. 1.—Showing percentage distribution of cases occurring each calendar month in the British Isles and Jersey. The numbers in brackets show the total number of cases of each type.
(Article by W. M. Allcroft.)

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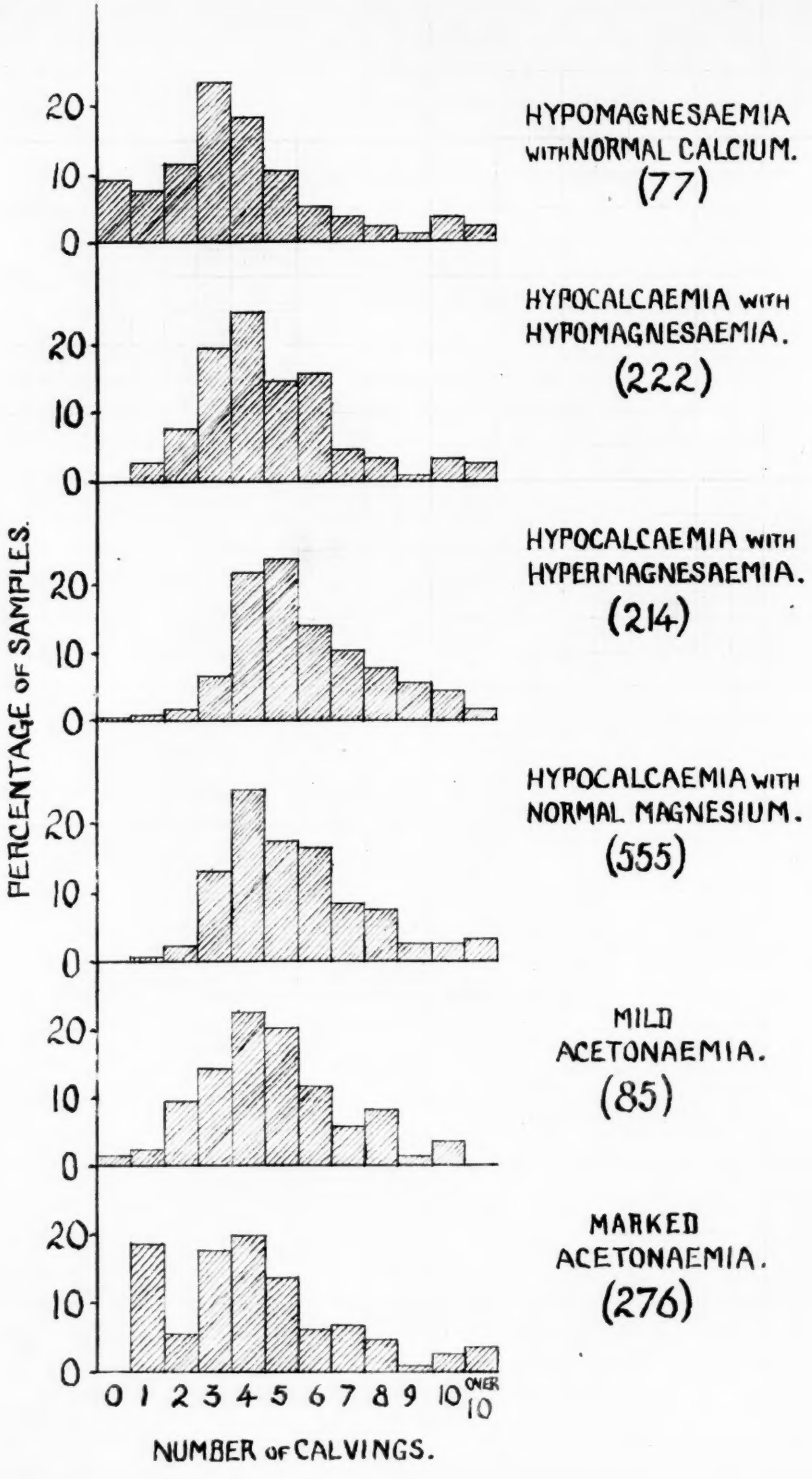

FIG. 2.—Showing percentage distribution of the occurrence of metabolic disorders at the different calvings. The numbers in brackets show the total number of cases of each type. (Article by W. M. Allcroft.)

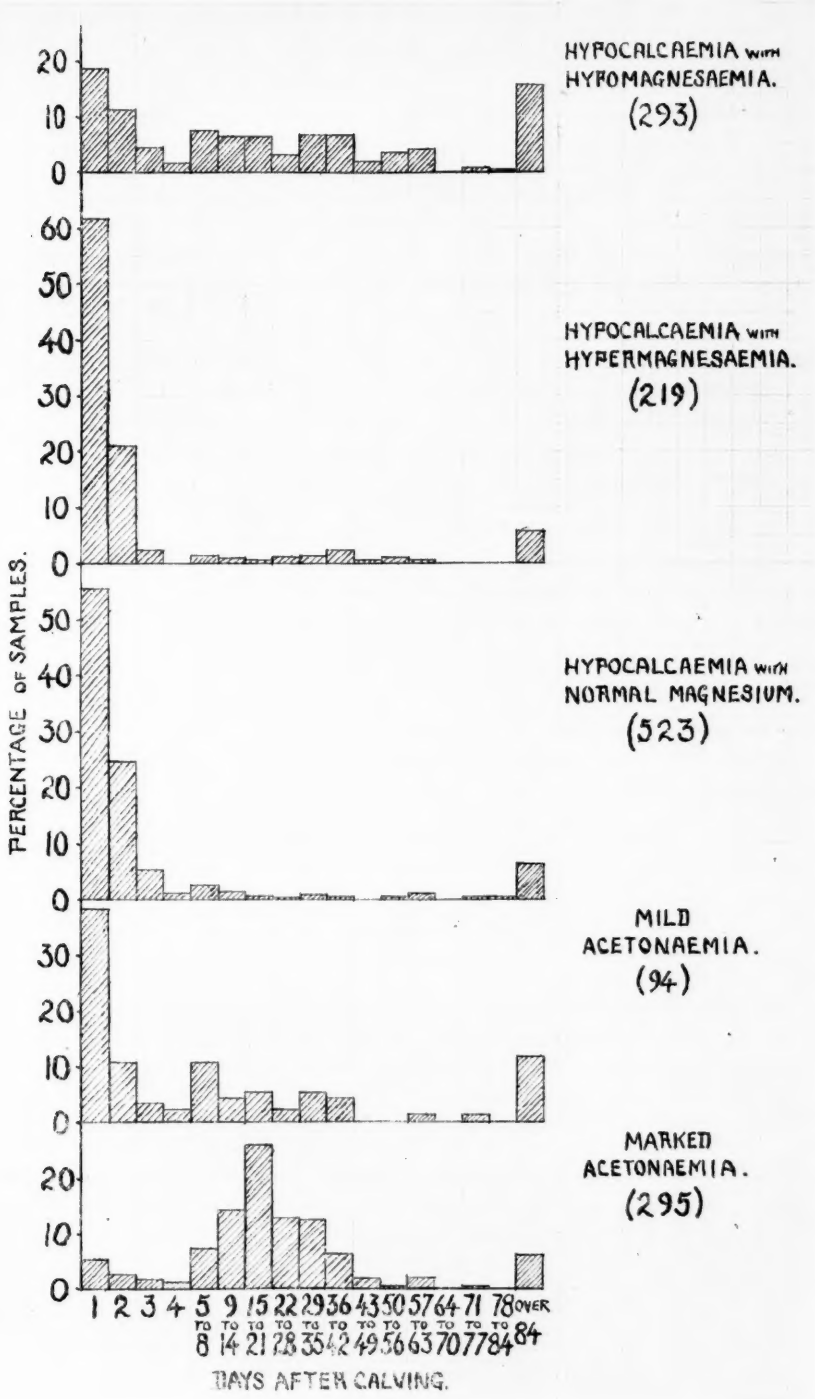
HYPOCALCAEMIA with HYPOMAGNESAEMIA. (293)
HYPOCALCAEMIA with HYPERMAGNESAEMIA. (219)
HYPOCALCAEMIA with NORMAL MAGNESIUM. (523)
MILD ACETONAEMIA. (94)
MARKED ACETONAEMIA. (295)
PERCENTAGE OF SAMPLES.
1 2 3 4 5 TO 8 9 TO 14 15 TO 21 22 TO 28 29 TO 35 36 TO 42 43 TO 49 50 TO 56 57 TO 63 64 TO 70 71 TO 77 78 TO 84 OVER
DAYS AFTER CALVING.

FIG. 3. Showing the percentage occurrence of cases at different times after the last calving. The numbers in brackets show the total number of cases of each type. The period after calving is divided into puerperium, interregnum and thereafter in weekly intervals up to 12 weeks.
(Article by W. M. Allcroft.)

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TABLE VI

Association of Certain Types of Biochemical Disorder with Time Interval since the Last Calving of the Cow

Number of days after last calving	Actual number of samples: Marked acetonæmia	Mild acetonæmia	Hypocalcæmia with normal magnesium	Hypocalcæmia with hypermagnesæmia	Hypocalcæmia with hypomagnesæmia	Hypomagnesæmia with normal calcium	All cases		Percentage distribution of samples: Marked acetonæmia	Mild acetonæmia	Hypocalcæmia with normal magnesium	Hypocalcæmia with hypermagnesæmia	Hypocalcæmia with hypomagnesæmia	Hypomagnesæmia with normal calcium	All cases
(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)		(i)	(j)	(k)	(l)	(m)	(n)	(o)
1	15	36	289	135	55	5	535	...	5.1	38.3	55.3	61.7	18.8	7.5	35.9
2	8	10	129	46	33	3	229	...	2.7	10.6	24.7	21.0	11.3	4.5	15.5
3	5	3	28	5	13	1	55	...	1.7	3.2	5.4	2.3	4.4	1.5	3.7
4	4	2	6		5	3	20	...	1.4	2.1	1.1		1.7	4.5	1.3
5-8	21	10	13	3	22	8	77	...	7.1	10.6	2.5	1.3	7.5	12.0	5.1
9-14	42	4	7	2	19	7	81	...	14.2	4.3	1.3	0.9	6.5	10.5	5.4
15-21	76	5	2	1	19	2	105	...	25.9	5.3	0.4	0.5	6.5	3.0	7.0
22-28	38	2	1	2	9	3	55	...	12.9	2.1	0.2	0.9	3.1	4.5	3.7
29-35	37	5	4	3	20	2	71	...	12.5	5.3	0.8	1.3	6.8	3.0	4.8
36-42	18	4	2	5	20	6	55	...	6.1	4.3	0.4	2.3	6.8	9.0	3.7
43-49	5			1	6	2	14	...	1.7			0.5	2.0	3.0	0.9
50-56	1		1	2	11	2	17	...	0.3		0.2	0.9	3.7	3.0	1.2
57-63	6	1	5	1	12	1	26	...	2.0	1.1	1.0	0.5	4.1	1.5	1.7
64-70						3	3	...						4.5	0.2
71-77	1	1	1		2	1	6	...	0.3	1.1	0.2		0.7	1.5	0.4
78-84			1		1		2	...			0.2		0.4		0.1
Over 84	18	11	34	13	46	18	140	...	6.1	11.7	6.5	5.9	15.7	27.0	9.4
								...							
Total	295	94	523	219	293	67	1,491	...	100.0	100.0	100.0	100.0	100.0	100.5	100.0
								...							

as that of marked acetonæmia. It rarely occurred in first calvers, but was often noted in milk fever cases.

Hypocalcæmia with Normal or High Magnesium

The first interesting point was the greater incidence of hypermagnesæmia in this series in cases from Jersey. That it is not a breed factor has been shown, and no other explanation can be offered, but speculation might suggest an association with climate or husbandry.

These disorders are essentially associated with the first 48 hours after calving. This factor probably accounts for the seasonal incidence, but in the absence of complete statistics of calvings it is not considered desirable to offer further comment. In Britain, both types occurred with similar seasonal incidence, 50 per cent. between May and August, and the rest fairly evenly spread over the other eight months. There were different incidences for the two types in Jersey, but no explanation is offered.

When the magnesium was normal, the third to sixth calving was the commonest time of occurrence, with greatest association with fourth calvings. In hypermagnesæmic cases, the fourth and fifth calvings were the commonest. In the earlier paper (Allcroft and Green [1934]), specific attention was drawn to the frequency of hypermagnesæmic cases, which had been noted by Sjollema and Seekles (1930) in Holland. In doing so now, it is only to add support to the view that milk fever is a pathological exaggeration of the physiological changes at normal calvings (Godden and Allcroft [1932] and Allcroft and Godden [1934]), when the calcium and inorganic phosphate were found to decrease and the magnesium tended to increase around the calving time.

Hypomagnesæmia, with Normal or Low Calcium

When hypomagnesæmia was found, 76.4 per cent. of the 406 cases showed concomitant hypocalcæmia, and of those cases of mixed hypocalcæmia and hypomagnesæmia, 36.2 per cent. were in the puerperium. Other than that association there was no indication of relationship to the time after calving. There was distinct seasonal incidence, which is of interest in connection with studies on seasonal hypomagnesæmia without clinical symptoms (Allcroft and Green [1938] and Allcroft [1947] in press). The association of hypomagnesæmia with the earlier calvings and with pregnant heifers is noted, but the only comment offered in explanation is that the disorder is often fatal and this would prevent cases recurring in older animals and so load incidence in favour of the earlier calvings.

Summary

Laboratory records of chemical analyses of blood and clinical reports received in connection with metabolic disorders of cows are presented and discussed.

Certain biochemically recognised types of disorder have been considered in relation to seasonal occurrence, the number of times the cow has calved and the time since the last calving.

The main tendencies are briefly discussed.

Acknowledgments

To Dr. H. H. Green, head of the Biochemical Department, for permission to use the general records of the diagnostic service; to Dr. A. Eden and Messrs. F. J. Salt and C. W. Clarke for assistance in the numerous analyses, and to the many veterinary practitioners who co-operated in the supply of material and information.

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THE SUSCEPTIBILITY OF SWINE FEVER-IMMUNE PIGS TO INTRACEREBRAL INOCULATION WITH *E. RHUSIOPATHIÆ*

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It is an established fact that pigs in general are refractory to artificial infection with *E. rhusiopathiæ* by the usual routes of inoculation. Many observers, Nocard and Leclainche (1903), Macé (1913), Hoare (1913), Ten Broeck (1920), Creech (1933), Kondo and Sugimura (1935), Schoening, Cochenour and Grey (1938), Watts (1940), Collins and Goldie (1940), and Breed (1940), have confirmed this fact in Europe, America and Japan.

Occasionally, whether as the result of unusually susceptible animals or of a particularly virulent strain of the organism, or for reasons to be here discussed, the infection may be successfully passaged a few times through pigs before it dies out. Having had little success over the course of years in infecting pigs with freshly isolated strains of *E. rhusiopathiæ* by the ordinary routes of inoculation, we decided to try the effect of intracerebral inoculation.

The pigs used in these experiments were bought direct from farms that were known to be free from any major disease. They were either large Whites or Saddle-backs, and varied in weight from 100 lb. to 250 lb. Some of them had previously been used at the Institute for potency tests of crystal violet swine fever vaccine.

The strain of *E. rhusiopathiæ* (Elmslie) selected for the intracerebral inoculations was of average virulence as judged by titration tests on mice. It

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invariably failed to kill pigs by subcutaneous or by intramuscular inoculation. A large dose (100 c.c.) of broth culture injected *per rectum* killed a three-months-old pig, but other pigs of the same age similarly treated remained normal. A six-month-old boar (No. 1,842) was killed by intratesticular inoculation, and a portion of spleen from this animal was freeze-dried and stored in vacuo at —20° C. Three years later tubes of serum broth were sown from this freeze-dried spleen and used as the inoculum for the first pig in the intracerebral series (Table I). The intracerebral inoculations were carried out under Nembutal anæsthesia.

Intracerebral Inoculations

Pig 3,417 was inoculated intracerebrally with 1 c.c. of a 24-hour serum-broth culture of *E. rhusiopathiæ* from the freeze-dried spleen of pig 1,842. Pig 3,417 died in forty-eight hours of acute swine erysipelas, and *E. rhusiopathiæ* was isolated in pure culture from the organs. It will be seen from Table I that fresh brain suspensions were used as the inocula for passages 3 to 10. Blood and suspensions of freeze-dried brain and spleen were also used as inocula (Table II).

Starting with fresh spleen suspension from P. 3.417 (2nd passage), the *E. rhusiopathiæ* infection was successfully passaged by intracerebral inoculation through a series of seven pigs, using fresh brain suspension in each case (see Table I).

The infection then apparently died out, as the two pigs (3,496 and 3,497) inoculated with fresh brain suspension from P. 3,462 (9th passage) showed only a brief period of dullness and inappetence and recovered. Two pigs (3,491 and 3,495) were then inoculated with a broth suspension of freeze-dried brain of P. 3,454 (7th passage), and these also showed only slight reactions and recovered; a fresh suspension of this brain had previously induced infection in P. 3,455 (8th passage). At this stage the histories of the twelve pigs that had been inoculated intracerebrally were examined. It was observed that of the eight that had died of *E. rhusiopathiæ* infection, seven were swine fever-immune pigs; and of five pigs susceptible to swine fever, four had either remained normal or had shown only a brief period of dullness and inappetence.

In the light of this information a second series of inoculations were carried out. Two pigs, 3,448 and 3,506, one swine fever-immune and one susceptible, were inoculated intracerebrally with a suspension of freeze-dried brain from P. 3,456 (6th passage). The immune pig, 3,448, died of acute erysipelas in 96 hours, whereas the susceptible pig, 3,506, showed only a slight reaction. It will be seen from Table II that fifteen more pigs, ten swine fever-immune and five susceptible, were inoculated; seven of the immune pigs died and three showed severe reactions and recovered, and of the five susceptible pigs, two showed severe reactions and three slight reactions, but none died. One swine fever-immune pig (No. 3,481—Table II) was infected by intracerebral inoculation of a suspension of spleen from a mouse that had died of *E. rhusiopathiæ* infection. Spleen suspension and blood from P. 3,481 were inoculated into five other swine fever-immune pigs (3,450, 3,484, 3,479, 3,483, 3,487); four of these were inoculated intracerebrally and one intravenously, and all died of acute *E. rhusiopathiæ*.

E. rhusiopathiæ was isolated in pure culture from all pigs shown in the tables as having died. An attempt was made to infect two swine fever-susceptible pigs

with *E. rhusiopathiæ* by giving a preliminary intracerebral injection of 2 per cent. starch in isotonic sodium chloride solution (Sawyer and Lloyd), and following this up with an intracranial inoculation of *E. rhusiopathiæ* culture (ex brain of P. 3,459, which died in 48 hours from an 1/C inoculation). Neither pig showed any reaction.

In view of these results, a re-examination was made of some earlier attempts to infect pigs with *E. rhusiopathiæ*. It was found that in one experiment, in which six boars, two swine fever-immune and four non-immune, had been inoculated intratesticularly, the two immune pigs died of swine erysipelas in 7 days and 48 hours, respectively, whereas the four non-immune pigs remained normal. We had failed to observe at the time that it was the swine fever-immune pigs only which had died, and the number infected (two out of six) did not appear to justify further investigation of the intratesticular route. The details of the experiment are as follows:—

Swine Fever-Immune Boar 1,842, six months old, was inoculated in the left testicle with 1 c.c. of a 24-hour broth culture of *E. rhusiopathiæ*. It became sick on the third day, temperature 107° C., both testicles swollen and numerous small purplish-red areas on the skin. Died on the 7th day. Spleen and blood cultures positive for *E. rhusiopathiæ*.

Swine Fever-Susceptible Boars 2,034 *and* 2,036, six months old, were inoculated intratesticularly with 1 c.c. of spleen suspension from P. 1,842. Both showed brief mild reactions.

Swine Fever-Immune Boar 1,847, seven months old, was inoculated intratesticularly with 1 c.c. broth culture sown from the spleen of P. 1,842. Died in 48 hours. *E. rhusiopathiæ* was recovered in pure culture from the blood, testicle and bone marrow.

Swine Fever-Susceptible Boars 2,037 and 2,038, six months old, were inoculated intratesticularly with 1 c.c. of the same material as used for P. 1,847. Both remained normal.

Summary

Two swine fever-immune boars and four non-immune boars were inoculated intratesticularly with *E. rhusiopathiæ*. The immune boars died of *E. rhusiopathiæ* infection in seven days and forty-eight hours, respectively, whereas of the four non-immune boars, two showed mild reactions and two remained normal.

Intracerebral Inoculations with E. rhusiopathiæ (First Series).

TABLE I.

Passage	Pig No.	Pig Immune or susceptible to Swine Fever	Route of Inoculation	Inoculum.	Result
2	3417	Immune	I/C	Broth culture ex spleen P.1842	Died 48 hrs.
3	3416	„	„	Fresh Brain Suspension	„ 96 „
4	3385	Susceptible	„	„ „ „	„ 96 „
5	3430	Immune	„	„ „ „	„ 48 „
6	3456	„	„	„ „ „	„ 72 „
7	3454	„	„	„ „ „	„ 72 „
8	3455	„	„	„ „ „	„ 72 „
9	3462	„	„	„ „ „	„ 72 „
10	3496	Susceptible	„	„ „ „	Lived
10	3497	„	„	„ „ „	„
8	3491	„	„	f.d.br. ex P.3454	„
8	3495	„	„	f.d.br. ex P.3454	„

f.d.br. = freeze-dried brain.
I/C = intracerebral.

TABLE II *(Second Series).*

Pig No.	Pig Immune or susceptible to Swine Fever	Route of Inoculation	Inoculum	Result
3448	Immune	I/C	f.d.br. ex P.3456	Died 96 hrs.
3506	Susceptible	„	f.d.br. ex P.3456	Lived
3383	Immune	„	f.d.br. ex P.3430	Lived—severe reaction
3466	Susceptible	„	f.d.br. ex P.3448	Lived—severe reaction
3492	„	„	f.d.br. ex P.3448	Lived
3571	Immune	„	Culture ex spleen P.1842	Lived—severe reaction
3499	„	„	f.d.br. ex P.3550	Lived—severe reaction
3481	„	„	Susp. mouse spleen	Died 48 hrs.
3450	„	„	Susp. spleen ex P.3481	„ 24 „
3484	„	„	Susp. spleen ex P.3481	„ 48 „
3479	„	„	Blood ex P.3481	„ 48 „
3483	„	„	Blood ex P.3481	„ 24 „
3493	Susceptible	„	Blood ex P3479	Lived—severe reaction
3539	„	„	Blood ex P.3479	Lived
3487	Immune	I/V	Susp. spleen ex P 3481	Died 24 hrs.

I/V = intravenously.

Observation I

To ascertain if the *E. rhusiopathiæ* invades the blood stream of swine fever-susceptible pigs following intracerebral inoculation.

Two swine fever-susceptible pigs, Nos. 3,468 and 3,469, were each inoculated intracerebrally with 1 c.c. of a broth suspension of spleen tissue from a pig (3,484) that had died of erysipelas.

Pig 3,468 showed considerable reaction and was killed at the 48th hour. *E. rhusiopathiæ* was recovered from the blood and spleen by culture and by mouse inoculation.

Pig 3,469 showed no reaction and was killed at the 72nd hour. *E. rhusiopathiæ* was recovered from the blood and spleen by culture and by mouse inoculation.

This observation shows that *E. rhusiopathiæ* invades the blood stream of swine fever-susceptible pigs following intracerebral inoculation.

Observation II

To ascertain if *E. rhusiopathiæ* invades the brain of swine-fever susceptible pigs following intravenous inoculation.

Two swine fever-susceptible pigs, Nos. 3,560 and 3,567, aged eighteen weeks, were each inoculated intravenously with 10 c.c. of a 48-hour broth culture of *E. rhusiopathiæ.*

Pig 3,560 was killed at the 24th hour. Cultures of the brain and blood were negative. Two mice inoculated with a suspension of brain died on the 4th and 8th day, respectively, and a culture of *E. rhusiopathiæ* was obtained from the heart blood and spleen in each case.*

Pig 3,567 was killed at the 48th hour. Cultures of the blood and brain were negative. One or two mice inoculated with blood died on the 4th day, and a culture of *E. rhusiopathiæ* was obtained from the heart blood and spleen. The

* We have not been able to find any reference in the literature to normal mice acting as "carriers" of *E. rhusiopathiæ*, and in view of their high susceptibility to that organism it is improbable that they do so. As a check, however, on the possible existence of latent infection, the organs of 50 of our stock mice were cultured for *E. rhusiopathiæ* with negative results. It would appear now to be generally accepted that *B. murisepticus* (Koch, 1878) and *E. rhusiopathiæ* (Pasteur and Thuillier, 1882) are identical.

two mice inoculated with brain suspension remained normal; and when tested later for immunity with *E. rhusiopathiæ*, both died.

Formation of Antibodies

The sera of five swine fever-susceptible pigs that had been inoculated intracerebrally with *E. rhusiopathiæ* and had either remained normal or shown only slight reactions, and the serum from one swine fever-immune pig (P. 3,383) that had been inoculated intracerebrally with *E. rhusiopathiæ* and had shown a severe reaction and recovered, were tested for protective antibodies. Each serum was injected intramuscularly into the left hind-leg of five mice in doses of 0.5, 0.4, 0.3, 0.2, 0.1 c.c., and at the same time a standard dose of virulent *E. rhusiopathiæ* was injected intramuscularly into the right hind-leg. All mice that received sera from the swine fever-susceptible pigs died, whereas only one mouse inoculated with serum (0.1 c.c.) from the swine fever-immune pig died. This result would suggest that protective antibodies are formed, following intracerebral inoculation, in those pigs only that undergo a systemic reaction. We have repeatedly observed that in pigs inoculated subcutaneously with a living, avirulent strain of *E. rhusiopathiæ*, which does not cause a systemic reaction, antibodies could not be demonstrated in the sera by mouse protection tests. Yet mice inoculated with the same living, avirulent strain of *E. rhusiopathiæ*, and in which no apparent systemic reaction occurred, always developed antibodies in their blood.

Discussion

The principal purpose of this work was to find a method of infecting normal pigs artificially with *E. rhusiopathiæ* so that the antigenic value of a living, avirulent strain of the organism that has been under study here for some years could be tested under controlled laboratory conditions. Field trials of an erysipelas vaccine, even on known infected farms, are unsatisfactory, because of the varying annual incidence of the disease, the relatively short erysipelas season in this country, and the considerable time necessary to obtain reliable data on the value of any new method of immunisation. The results obtained were unexpected and are, perhaps, worth recording. It would appear from the small series of experiments here described that a high percentage of pigs immunised against swine fever (crystal violet vaccine, followed by virus) can be infected by inoculation of either cultures of *E. rhusiopathiæ* or of tissues (brain, spleen or blood) from erysipelas infected animals. Pigs susceptible to swine fever appeared, on the contrary, to be strongly resistant to inoculation with similar material.

Out of a total of nineteen swine fever-immune pigs inoculated intracerebrally with *E. rhusiopathiæ*, sixteen (84 per cent.) died of swine erysipelas and three showed severe reactions and recovered; whereas out of 14 swine fever-susceptible pigs, only one died, five showed severe reactions, while the remainder showed either slight reactions or remained normal. The interval between the subcutaneous inoculation of swine fever virus and the intracerebral inoculation of *E. rhusiopathiæ* was never less than thirty days, and sometimes considerably more. It was clear that swine fever virus was not directly responsible for the deaths that followed the intracerebral inoculation of *E. rhusiopathiæ*, as if that were so the

deaths would have occurred among the swine fever-susceptible pigs and not among the swine fever-immune pigs. Twelve swine fever-susceptible pigs were inoculated, ten intracerebrally and two intratesticularly, with suspensions of brain, blood or spleen obtained from swine fever-immune pigs and no evidence of swine fever appeared in any of these pigs during the period of observations, which was never less than thirty days.

As swine fever virus is pantropic and can be recovered from the brain of naturally or of artificially infected pigs, it was at first considered that the susceptibility of swine fever-immune pigs to intracerebral inoculation with *E. rhusiopathiæ* might be related to a preliminary weakening or damaging of the brain by the virus, but this view was upset by the successful infection of three swine fever-immune pigs, two (1,842, 1,847) by intratesticular inoculation and one (3,387) by intravenous inoculation. Furthermore, it has been shown (Observation I) that the intracerebral inoculation of swine fever-susceptible pigs with *E. rhusiopathiæ* is followed by infection of the blood stream, just as occurs in swine fever-immune pigs.

Speransky (1935) investigated the influence of the nervous system on the pathogenesis of disease, including those caused by specific agents. He postulated that the nervous system is an organ which cannot be altered locally without affecting the entire system, and that disturbance of the nervous pattern influences the pathogenesis and symptomatology of disease processes.

The intracerebral inoculation of swine fever-immune pigs with *E. rhusiopathiæ* was, in the majority of cases, followed rapidly by an acute erysipelas bacteræmia, with death usually occurring in from 24 to 72 hours. In some animals typical local areas of reddish-purple discolouration extended over large areas of the body.

It is an interesting fact, although it may be merely a coincidence, that in countries in which active immunisation against swine fever (serum-virus method) is extensively practised, swine erysipelas appears to be widespread, and in some areas it has become a major disease, second in importance only to swine fever. In Great Britain, where no method of active immunisation against swine fever has ever been practised, swine erysipelas is mainly of the chronic type, and although the disease is of considerable economic importance, it has shown no apparent tendency over the course of years to spread outside of well-defined areas or to assume a more virulent form. There are, of course, other factors, such as diet, soil, climate and density of pig population, that probably exercise considerable bearing on the epizootiology of the disease, and as these vary in different countries it is not possible to make any reliable comparison. It may be, however, that the reaction (vaccination shock and leucopenia) caused by the serum-virus method of immunisation is sometimes a contributory factor to the incidence of swine erysipelas, and might even be the "provocative factor" referred to by Breed (1942). That there are other predisposing causes is beyond question, as is proved by the resistance exhibited by pigs of all ages to artificial infection with recently isolated virulent strains of *E. rhusiopathiæ*. Swine erysipelas is indeed a good example of a disease in which the predisposing causes would appear to be equally important as the exciting cause.

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Further investigation will be necessary to determine whether the susceptibility of swine fever-immune pigs to artificial infection with *E. rhusiopathiæ* has any bearing on the pathogenesis of the disease under field conditions. Observations on this point cannot be made in this country, as active immunisation against swine fever, involving the use of live virus—apparently an essential factor—is not carried out. We have not yet ascertained the duration of susceptibility of swine fever-immune pigs to *E. rhusiopathiæ*, or whether the degree of reaction caused by immunisation against swine fever has any influence on their subsequent susceptibility. It would appear from some small preliminary experiments that it might be possible to devise a method for assessing the protective value of swine erysipelas vaccine by tests on swine fever-immune pigs.

Conclusion

A high percentage of pigs actively immunised against swine fever (crystal violet vaccine, followed by virus) were found to be susceptible to intracerebral inoculation of either cultures of *E. rhusiopathiæ* or of tissues (brain, spleen or blood) from erysipelas infected animals. Swine fever-immune pigs appeared also to be susceptible to intratesticular and intravenous inoculation with *E. rhusiopathiæ*. Pigs susceptible to swine fever appeared, on the contrary, to be strongly resistant to intracerebral inoculation with similar inocula.

Acknowledgment

To Mr. E. C. Wright for valuable assistance and for designing a table for restraint of the pigs during the intracerebral inoculations.

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"ROUND HEART DISEASE" IN POULTRY

By D. LUKE, B.Sc., M.R.C.V.S.
Stormont, Belfast.

DURING the last few years considerable losses among poultry flocks in Northern Ireland have been observed, particularly among birds 3-7 months old. The disease condition is characterised by sudden death. The outstanding abnormality noted at post-mortem examination of birds which have died from this disease is to be found in the heart, and the condition has been designated "Round Heart Disease" on account of the peculiar change in the contour of that organ.

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The author has been unable to find any references in the literature to a similar condition.*

At the moment, 36 outbreaks of "Round Heart Disease" are under review, and some interesting features of the disease are recorded here. Typically preliminary symptoms are absent. In only one outbreak out of the 36 did the owner record any symptoms, and here it was noticed that the birds which subsequently died were dull and disinclined to move for about two hours before death. In all the other outbreaks death occurred with dramatic suddenness. Birds may be found dead under the perch or seen to topple over in the run. In a typical history deaths occur commonly at feeding time, and birds running up at the sight of the feed bucket suddenly heel over, dead. In some cases affected birds lie paddling with their feet for a short time before death. In the majority of cases, however, apparent normality and death are only separated by a few seconds. The suddenness of death often makes an owner suspect some form of poisoning. Birds which have died from "Round Heart Disease" are always in good condition. There is no general setback in the flock during the period when deaths are occurring, and the survivors are consistently reported as doing well and even above average for their age.

Deaths occur over a period of 3-4 months and then cease. The daily death rate seldom exceeds 1-2 birds. The disease does not seem to be confined to any one breed. It does not appear to spread from the particular flock in which it is first seen, though we have had one instance where deaths from "Round Heart Disease" occurred in Leghorns and Rhode Island Reds of similar age in the same run.

In the series of outbreaks under review deaths occurred between early June and November. In the 36 outbreaks 481 deaths occurred among 3,214 birds. In 10 of the outbreaks deaths began when the birds were 3 months old, in 10 outbreaks at 4 months, and in 4 outbreaks each at 5 months, 6 months, 7 months, and between 8-18 months, respectively. (See table.) We have not seen the disease in birds over 18 months old, and in only 2 outbreaks did the deaths begin at over 12 months. The disease has been seen mainly in young pullets, but we have had three cases in young cockerels. We have not been able to corelate the presence of "Round Heart Disease" with the existence of any other disease in the affected flocks, e.g., Fowl Paralysis, etc., and our post-mortem findings have, up to the present, been exceptionally uniform. (See under.)

In 17 of the outbreaks the birds were bought as day-old chicks from various hatcheries in Northern Ireland. Thirteen outbreaks occurred in birds hatched from eggs derived from private breeders, and in six cases the birds were hatched from eggs from the breeding flocks on the farms where the "Round Heart Disease" occurred. The disease has been known to occur on the same farm in two successive years. The average death rate in the present series of outbreaks was 14.9 per cent., with individual flock mortality varying from 2 to 54 per cent. It is probable that this percentage mortality errs on the low side, as in some cases at least the statistics were collected before all the deaths had occurred.

* Since this article was written the author's attention has been drawn to a paper, "Enzootic Fatal Syncope (Toxic Heart Degeneration) of Fowl," which appeared in the *Australian Veterinary Journal* (1946), *22*, 144-149. Time has not yet permitted the comparing of the two conditions.

Age at which deaths began.	No. of deaths in each age group.	No. of outbreaks in each age group.	No. of birds in affected flocks.	Percentage death rate.
3 months ...	140	10	792	18.6
4 ,, ...	135	10	873	15.4
5 ,, ...	69	4	364	18.9
6 ,, ...	34	4	260	13.0
7 ,, ...	59	4	285	20.2
8-18 ,, ...	37	4	640	5.8

Three outbreaks occurred in June, 6 in July, 7 each in August and September, 9 in October, and 4 in November. From the figures given above it will be appreciated that the disease is a serious one, especially among young growing stock. There appears to be a definite age susceptibility and a seasonal incidence, though the latter may be more apparent than real, as the poultry population within the susceptible age limits is greatest during the period when the death rate was highest. Critical observation over an extended period should provide more information on this point.

The disease is at present under investigation, and the following notes on post-mortem findings are recorded for the information of others who may have experience of this or a similar condition. Without exception, all the birds examined post-mortem were in good condition. The pectoral muscles are usually pale. The most striking change is seen in the heart, which is enlarged, bluish-pink in colour, and the muscle seems spongy. The typical tapering contour of the organ is lost and the ventricles have a rounded outline, with the apex invaginated in a proportion of cases. The superficial vessels of the heart are markedly congested. In some cases the heart is encased in a yellowish, jelly-like clot. The interior of the heart appears normal to a naked eye examination. There is usually a marked excess of pericardial fluid. Excess abdominal fluid is a common feature and an œdematous condition of the lungs has been seen. In several specimens a jelly-like clot was found over the ventral surface of the liver. The condition of the liver is not constant; in some it is much congested and in others it is paler than normal. The spleen is usually normal in size, colour and consistency, though in a few cases some slight enlargement has been noted. The kidneys appear normal and nothing unusual has been seen either in the intestine or in its contents. In a high proportion of cases the mucous membrane of the throat has a bluish colour, and some catarrh of the larynx and trachea has been noted. The primary lesion is undoubtedly cardiac, and the various other changes are probably secondary to the failing circulation. One of the most striking features of the disease is the extent of macroscopic change found in the heart muscle without the bird apparently showing any preliminary symptoms of circulatory embarrassment.

Cultures on all the usual laboratory media have been made from the heart, heart blood, pericardial fluid, liver, spleen, tracheal mucous membrane, intestines and kidneys, and grown aerobically and anaerobically: these have consistently failed to show anything of pathological significance.

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Summary

A serious disease condition characterised by sudden death and causing considerable mortality in young poultry in Northern Ireland is described. It is designated " Round Heart Disease " from the characteristic change in the contour of the heart consistently noted at post-mortem examinations. The losses in 36 outbreaks of the disease are reviewed and analysed.

CLINICAL ARTICLES

A CASE OF CÆSARIAN SECTION IN THE BOVINE

By PETER KIDDLE and E. W. JONES

Department of Surgery, Royal Veterinary College

Subject

A PEDIGREE Guernsey Heifer, Primipara, aged 2½ years (" Normandy Belle ").

History

The animal was first seen on September 16, 1946, when parturition was reported to be six days overdue. Straining was marked and upon examination the cervix was found to be tightly closed. The fœtus was normally presented and no fœtal movements could be detected. Examination on September 17 revealed a closed cervix and straining had almost ceased. Twice daily visits were made till September 19, when the cervix was only sufficiently dilated to admit two fingers. Cæsarian section was thought to be indicated, and the heifer was sent to the College Hospital.

Examination

The heifer was admitted on September 19. The temperature, pulse and respiration were normal; the animal was bright and ruminating, straining was absent.

Vaginal exploration revealed a completely relaxed vagina, but the cervix would only admit three fingers. The uterine seal had broken down and there was no uterine discharge.

Rectal exploration revealed an anterior presentation. No definite conclusion could be made as to the viability of the calf; feeble movements of the fœtal limbs could be detected, but it was difficult to be certain that these did not coincide with ruminal movements. No ocular movements were appreciated and no response could be obtained by placing the fingers in the calf's mouth.

Absence of straining suggested some degree of uterine inertia, and it was felt that a waiting policy would not improve what appeared to be a good surgical risk. Consequently, an immediate Cæsarian operation was decided upon.

Operation

Anæsthesia and Restraint: 1 oz. of chloral hydrate with ¼ oz. sodium citrate in 750 c.c. of sterile distilled water was injected intravenously. The animal

assumed recumbency and was restrained in the right lateral recumbent position, with legs extended. Surgical anæsthesia was obtained by chloroform inhalation, using an initial dose of ½ floz. followed by ¼ floz. doses when required, a total of 9¾ flozs. was administered during the course of the operation.

Preparation of Site: It was decided to make a left paramedian incision, about four inches lateral to the mammary vein, just within the fold of the flank. A large area around this site was clipped, thoroughly cleansed with soap and water, and sprayed with a saturated solution of iodoform in ether. Sterile cloths were then applied.

Technique: An incision, approximately fourteen inches long, was made through skin, panniculus, the aponeuroses of the external and internal oblique, transversalis muscles and the peritoneum. The little hæmorrhage encountered was controlled by forci-pressure. Upon entering the abdominal cavity, the omentun was reflected forwards and the uterus was brought up into the incision and held there by one of us (E.W.J.). A longitudinal incision was then made in the uterine wall of approximately eleven inches, care being taken to avoid cotyledons. Forci-pressure was again sufficient to control hæmorrhage. The membranes were incised, both hands were inserted into the uterus. Owing to the atonic state of the uterus, the anterior extremity of the calf, which is normally located just within the pelvic cavity, was lying under the incision. The forelegs were grasped, brought into the incision, and the calf carefully eased out to prevent tearing of the uterine wound.

The incised edges of the uterus were held just outside of the skin incision to prevent spilling of the uterine contents into the peritoneal cavity. By depressing the ventral margin of the skin incision, the fœtal fluids were drained to the exterior. Placental separation had not commenced and some difficulty was encountered in removing the fœtal membranes. The uterine incision was closed by a double layer of Lembert sutures, an inner continuous layer, followed by an interrupted layer, using No. 3 twenty-day chromic catgut. Whilst the second row of sutures was being inserted, 50 units (5 c.c.) posterior pituitary extract was given intravenously into the posterior auricular vein, consequent upon which the uterus contracted down so quickly that some difficulty was experienced in inserting the last few sutures.

The abdominal incision was then closed, and even though there was very little tympany of the rumen, there was much gaping of the wound edges. In order to prevent tearing of the peritoneum, it was found necessary to include muscle, aponeurosis and peritoneum together in a single layer of interrupted sutures, using No. 5 twenty-day chromic catgut. Further support to the muscle incision was supplied by eight eversion sutures, using half-inch tape, which included muscle layers and skin. Iodoform and ether was sprayed over the edges of the incision and the animal was moved on to her sternum.

Calcium borogluconate (4 oz.) with glucose (3 oz.) in 500 c.c. of sterile water was given intravenously. The subject was standing within 30 minutes.

Calf

A small Guernsey bull calf weighing about 55 lb. was delivered. It was only after 20 minutes of artificial respiration that breathing became spontaneous;

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carbon dioxide and prussic acid did not appear to be of any benefit. Throughout the period of artificial respiration, considerable amounts of frothy mucus exuded from the nostrils. Normal respirations having commenced, the calf was thoroughly rubbed down and kept warm overnight, and was quite normal and vigorous the following morning. It proceeded to put on weight rapidly during the time it was in hospital.

Post-operative Course

During the first 48 hours after operation, the temperature rose to 102.5° F.; the appetite was good and general appearance was bright. There was some pain and localised œdema at the site of operation. At first the heifer resented the mere presence of the calf, but grew very attached to it by the end of a week. Towards the end of the second day, the temperature began to rise and had reached 104.8° F. by the morning of the third day, when the respirations were accelerated (55) and mainly of a thoracic nature. The pulse was frequent but strong (85 a minute). The fæces were reduced in quantity and there was evidence of pain on defæcation. The appetite was not good, but at no time during the period of convalescence did the animal completely refuse to eat, and she continued to give more than enough milk to feed the calf.

Examination per vaginum revealed a slight blood-stained mucoid discharge, but no evidence of pus. Penicillin was injected subcutaneously in doses of 30,000 units every four hours for four days, but no marked result was obtained and the temperature continued to fluctuate between 103 and 104.5° F.

At the end of this period, the penicillin was discontinued and the administration of sulphanilamide in electuary was commenced (3 oz. followed by 1½ oz. every eight hours for five days). The appetite gradually improved, although the temperature remained high (103-104° F.). The vaginal discharge had developed a dirty brown colour and a foul odour, and as rectal examination revealed the presence of pus within the uterus, 50 units (5 c.c.) of posterior pituitary extract was given intramuscularly every other day until the subject was discharged thirteen days after the operation.

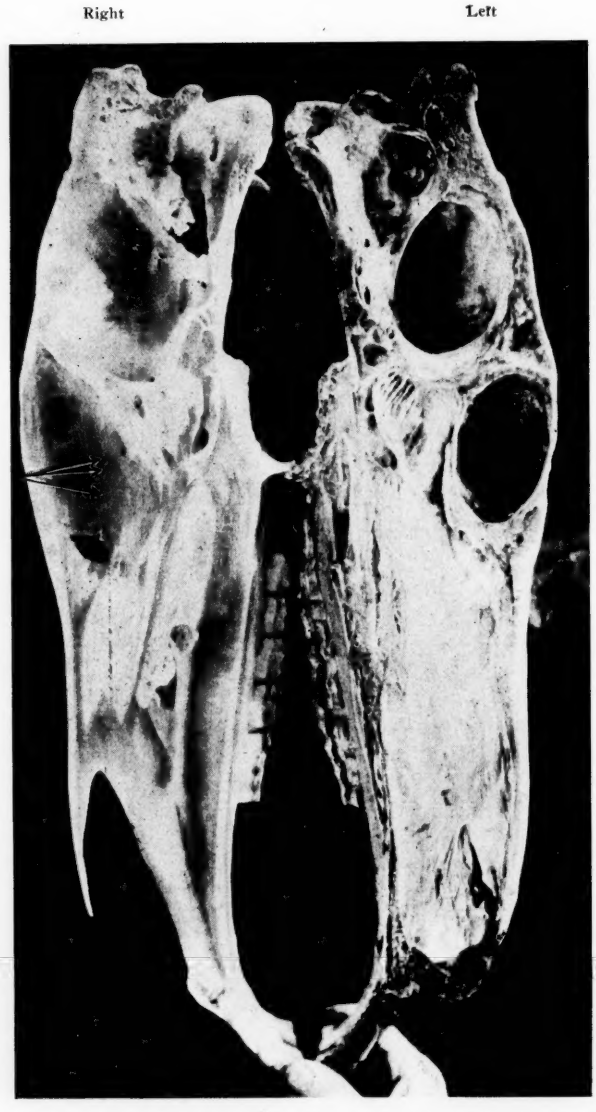
Details of progress after operation were supplied by Mr. F. E. Rickard, M.R.C.V.S., of Woking. Two days after discharge from hospital, the sutures were removed, first intention healing having occurred. Some pus drained from the suture holes for ten days. Pituitrin was administered twice weekly for three weeks, by which time the uterus had contracted down to a size rather less than twice normal size. The heifer gradually improved in general health and her milk yield increased to 2½ gallons daily three weeks after discharge.

Discussion

The cause of dystokia is not clear. In view of the fact that the subject was primipara, it is improbable that the primary lesion was in the cervix. The uterus was very atonic at the time of operation and the cause may have been primary or early secondary uterine inertia.

The difficulty in resuscitation of the calf may have been in large measure due to chloral hydrate, and this may be, like Nembutal in the bitch, a contra-indication for the anæsthetic.

PLATE IV



An unusual cavity in the frontal region of a horse.
By MISS MORANT.

The fact that penicillin had no effect on the temperature may have been in part due to inadequate dosage. Watts and McLeod have shown that at least 500,000 units every three hours must be given s/c to produce an effective bacteriostatic level in the blood of the bovine.

Since compiling this case report a letter has been received from the owner [received November 25, 1946]. The heifer is doing well, and has been giving 2½ gallons of milk per day for the last four weeks. The calf has grown and looks very promising.

Acknowledgments

The authors are greatly indebted to Professor Formston, F.R.C.V.S., for encouragement and advice in preparation of this case report, and to F. E. Rickard, M.R.C.V.S., of Woking, for detailed information of the case whilst under his supervision.

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P. S. Watts and D. H. McLeod: *J. Comp. Path. and Therapeutics*, Vol. 56, No. 3, page 170.

AN UNUSUAL CAVITY IN THE FRONTAL REGION OF A HORSE

By Miss K. MORANT, M.R.C.V.S
Shenley

Subject

A 15-YEAR-OLD thoroughbred mare.

The mare was in regular use at a local riding school and in good general health and condition. For two years a small swelling had been noticed which was situated in the midline of the head, just below the level of the supra-orbital foramina. This swelling gradually increased in size up to that of a small walnut. It was painless and fluctuated on pressure. Beneath the swelling one could feel a hole in the bone, somewhat resembling a fontanella and about the size of a shilling. There was no nasal discharge.

Dr. McCunn saw the case with me and he was of the opinion that it was not an ordinary sinus case, but probably some congenital cystic state. Under local anæsthesia the skin was incised and the cavity opened. It contained about half a pint of a clear serous type fluid.

The membrane lining the cavity, which was approximately the size of a large orange, appeared to resemble normal frontal sinus membrane, but there was no connection with the frontal sinus. At the base of the cavity there was a small valve-like hole which appeared to communicate with the right maxillary sinus. The tip of the finger could be inserted through this hole. It was enlarged slightly by the finger. The cavity was washed out and a little of the irrigating fluid drained through the nose. It was hoped that this drainage would increase and so prevent refilling. This did not happen; the membrane became infected and the cavity filled with a pus-like fluid. Swabs were taken and examined by Dr. Hare, who reported positive B.H.S. It was then packed with gauze soaked

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in " Pennoten," the latter being renewed every other day for 14 days. Also, 30 c.c. of Vivicillin was injected intramuscularly on two occasions with a 24-hour interval.

The nature of the discharge altered to a thick yellow substance with an offensive odour after the second Pennoten dressing.

A vaccine was prepared and 5 c.c. injected every fourth day, and there was now a slight nasal discharge. The discharge gradually decreased in quantity but remained chronic, resembling an odourless nasal catarrh. The breathing was not affected, but on exercise a slight frothy discharge issued from the original hole. The condition appeared to be gradually clearing up, but an attack of enteritis supervened and the animal was destroyed. At post-mortem examination, the skull was sectioned in the longitudinal midline. A large ovoid cavity, 3½ x 3 in., was found which extended from the level of the supra-orbital foramina towards the nose and over the superior turbinate bone. Laterally, it extended to within 1¼ in. of the orbital rim.

It was lined by a membrane similar to that of the sinuses and contained a small quantity of serous-like fluid. The original midline hole was filled in by a strong fibrous tissue membrane. In the right ventral part of the sinus there was a small hole which communicated with the right maxillary sinus. This hole admitted the handle of a scalpel. There was no communication with the frontal sinus. The frontal and maxillary sinuses appeared to be healthy.

I am of the opinion that this sinus was probably of congenital origin, that the serous fluid secreted therein had for many years drained away via the right maxillary sinus, and probably the distention of the cavity was due to some local swelling of the membrane lining the aperture. From the appearance of the hole in the forehead region, one would suspect that it was also of congenital origin, yet it is strange that it passed unnoticed for many years until the collection of fluid in the interior caused the skin over the hole to bulge. One half of the skull was boiled out in order to demonstrate the site of the small drainage hole. The other half was macerated carefully in order to preserve the lining membrane. The boiled out specimen shows that there is a thin bony septum between the cavity and the maxillary sinus, and at the right lateral periphery of this septum there are a few foramina communicating with the maxillary sinus. Part of the septum bulged into the medial section of the maxillary sinus.

ABSTRACT

NERVOUS DISEASES OF ANIMALS

By C. F. SCHLOTTHAUER

THIS very useful and well-documented official report,* the first since April, 1942, from the Committee of Neurology to the meeting of the American Animal Hospital Association covers only selected areas of the wide field of veterinary neurology (research data on rabies being left to another speaker). The chief items are :—

(1) Virus diseases : Equine encephalomyelitis, extremely widespread in the U.S.A. before an effective vaccine had been prepared, is now rarely seen. The

* *The North American Veterinarian*, 27, No. 11 (November, 1946), p. 696.

disease still occurs sporadically in various domesticated and wild animals, including birds, and in man.

Of the four strains of equine encephalomyelitis virus originally reported, the eastern, western, Venezuelan and Columbian strains, the two latter have, by mouse protection tests, been found to be immunologically identical.

Mosquitoes are considered to be the common vectors, but certain other parasites and possibly fowls may be reservoirs of the infection. It has been shown by blood precipitation tests that out of 788 mosquitoes caught in the wild state, 209 had fed on fowls, 198 of them belonging to the two species, ***Culex tarsalis*** and *pipiens*, regarded as the chief vectors of the disease. The dog tick, ***Dermacentor variabilis*** *Say*, and the common chicken mite, *Dermaryssus gallinæ*, also contained the virus and, therefore, are possible vectors.

In Portugal the occurrence of encephalitis in horses, cattle, sheep, swine, cats, and fowl has recently (1943) been reported. Viruses were isolated from cases of disease in all these species. The mammalian viruses were pathogenic to guinea-pigs, rabbits and pigeons, whereas fowl viruses were pathogenic for pigeons only.

Immunological comparisons (complement fixation, serum neutralisation and cross-resistance tests) revealed that the viruses of Russian encephalitis and louping-ill form one closely related group. The viruses of St. Louis and Japanese B encephalitis form another less closely related group. The western equine encephalomyelitis virus was distinct from the others.

The routes of entry of the viruses into the nervous system from the blood stream were studied by intravenous and intra-arterial injections of the viruses of pseudorabies, herpes and infectious myxomatosis into rabbits. In some instances drugs known to increase the permeability of the blood-brain barrier and particles suitable to produce intravascular emboli were added to the injected material. It was found that the viruses of herpes and pseudorabies first infect the viscera and from there ascend to the spinal cord, thence to reach other parts of the CNS. These observations may account for the early clinical symptoms of spinal affection in animals which later develop encephalitis. Virus may also reach the olfactory bulbs from the nose. Myxomatosis virus reaches the CNS directly via the blood stream. On embolisation herpes virus may also cause primary encephalitic infection. The supposed increase in membrane permeability had no appreciable effect.

Racoons have been found susceptible to the virus of fox encephalitis. Microscopical examination for the presence of inclusion bodies of the lymph nodes draining the site of inoculation is a ready means of detection of the infection, but most satisfactory is the examination of the cornea after intra-ocular injection.

(2) Hysteria and epilepsy: The experimental evidence is reviewed, which seems to support the assumption that certain diets can induce the occurrence of fits in dogs. Some investigators ascribe the actual outbreak of fits in susceptible animals to dietary deficiencies, such as lack of thiamin or lysine or some other specified amino acids present in raw casein but absent in some commercial food preparations, baked or heated (the addition of thiamin, choline and pyridoxone in these cases being without curative value).

Other authors regard the fits as a reaction to toxic substances, such as gliaden or gluten, both being plant proteins. On addition of 10 per cent. gluten, an

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Reproductive Disturbances of Cattle and Their Treatment (Die Fortpflanzungsstorungen des Rindes und deren Behebung). By PROFESSOR W. HOFMAN, University of Berne, with Foreword and Appendix by Professor G. Flückiger, Director of Veterinary Services. Verlag Hans Huber, Berne, 1945. Price, Swiss Fr. 4.80; pp., 86; 16 photographs and nine coloured plates.

The aim of the writers of this booklet has been to produce a short but comprehensive and easily understood guide to the physiology of reproduction and the disorders thereof in cattle. It is intended for the use of agricultural students taking courses in animal reproduction, but contains also much useful advice to veterinarians and others connected with animal husbandry.

The text of the booklet is divided into four sections, each dealing with some aspect of disturbed reproductive function, viz.: (i) dealing with ovarian function and the effects of malnutrition, mineral deficiency and high milk yield; (ii) treats in a general way certain infections of the reproductive tract, including infection of the Fallopian tubes, trichomoniasis, brucellosis, contagious granular vaginitis and tuberculosis. It is interesting to note that Swiss workers still relate ovarian cysts and persistent corpora lutea to infection in the tubal parts of the genitalea: (iii) deals first with fœtal abnormalities and then proceeds to a discussion of infertility in both male and female animals. Under "infertility in the bull" the following conditions are dealt with: absence of libido in young bulls, lazy bulls, various diseases and abnormalities of the internal and external genitalia, traumatic injuries to the testes, and inability to thrust. Under "infertility in the female" the discussion is mainly concerned with various infections of the reproductive tract. Reference is also made to inherited malformations of the reproductive organs of breeding stock: (iv) is a consideration of some of the causes responsible for abnormal reproduction in bulls, e.g., overuse and bad costal behaviour.

The Appendix by Flückiger takes the form of notes and comments on reproductive disturbance, and includes such topics as: theoretical maximal reproductive ability of the cow; reproductive index; reduced fertility; economic losses; and control measures. In the Appendix are also included details of Swiss legislation in connection with artificial insemination; the control and prevention of brucellosis, including vaccination with "Buck 19" (? ? Strain 19 of U.S. Bureau of Animal Industry), and tuberculosis eradication.

Much of the information given in this booklet will be familiar to veterinary practitioners in Britain, and no new methods of diagnosis or treatment will be found in the text. This is, of course, to be expected, since the author's primary aim seems to have been to provide a practical guide-book for the livestock breeder. For anyone specially interested in bovine sterility, the booklet is worth possessing on account of its excellent illustrations, particularly for the high standard of the coloured plates. N.J.S.

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